PERIPHERAL FACIAL DIPLEGIA AND PALATAL INVOLVEMENT.*

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Facial diplegia in its complete form has always excited the interest of the observer, not only on account of the comparative rarity of its occurrence, but also on account of the striking clinical picture which it presents and of the distress and inconvenience which it causes the patient.

Since Charles Bell, in 1836, first made known to us this clinical picture, the subject has been studied in its entirety by Davaine[1], Wachsmuth[2], Pierreson[3], and Sainton[4], while very many other observers have given us reports of single instances. Indeed so many single instances have been recorded and Sainton has so recently given such a complete and excellent description, with full literary references, that it would be superfluous to publish still more personal observations were it not for the fact that observers still differ as to the presence or absence of certain symptoms. Nor would this alone induce me to report the four cases which I have observed were it not for the fact that in my opinion their study adds weight to our present position in regard to the nerve supply of the soft palate.

In such a study we must bear in mind that facial diplegia may occur in diseases of the pons and medulla oblongata, as part of a multiple palsy of cerebral nerves (Rad[5]), in the course of cephalic tetanus, as well as, and particularly in, the course of a multiple neuritis (Pierreson, Eisenlohn, Strümpell[6], Starr[7], and others), due to rheumatism (Romberg, Mott, Stintzing[8],

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[1] Davaine, C. M. Gazette médicale de Paris, Nos. 46, 48, 50, 1852; Nos. 2, 3, 1853.

[2] Wachsmuth, Adolf. Monograph. Dorpat, 1864, Part II., pp. 81-114.

[3] Pierreson. Arch. génerales de médecine, August, September, 1867, Vol. X., pp. 159-296.

[4] Sainton, M. Paul. Gazette des hôpitaux, 1901, p. 1265.

[5] Rad, C. Von. Zeitschrift f. Nervenheilkunde, Vol. XVII., 1900.

[6] Strümpell, Ad. Neurol. Centralblatt, 1889, No. 21.

[7] Starr, M. Allen. Organic Nervous Diseases, 1902.

[8] Stintzing. Munehene med. Wochenschrift, January, 1893, Nos. 1, 2.

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Raymond), alcohol (Oppenheim[9]), influenza (Barkas[10]), or any other toxic or infectious cause.

Only such cases as are certainly peripheral in their origin are serviceable for casting any light upon the question, and of these, only those in which clinically the dominant symptoms are those of the facial diplegia. The supranuclear cases and the nuclear, or bulbar ones, cannot be utilized in this connection.

The nerve supply of the palate has been the cause of much discussion. In 1852 Davaine, writing upon facial diplegia, stated that the majority of anatomists believed the nerve fibers to the levator palati and azygos uvulæ muscles to be derived from the facial through the connection of its trunk with the Vidian by the petrosal nerves. Debron[11] had, about this time, by means of galvanic experiments upon dogs, shown that the nerves to the soft palate did not go from the seventh, but from the glossopharyngeus directly, and Davaine, in order to explain this divergence of views, endeavored to show that the fibers from the glossopharyngeus should be looked upon as sensory and those from the facial as motor fibers of the soft palate, and that therefore paralysis of the palate, when it occurred, was actually caused by affections of the facial.

From 1866, when Longet cast the weight of his authority into the scales, it had been accepted as a fact that the facial nerve governed the contraction of most of the muscles of the soft palate and that this motor influence was effected in the manner already mentioned.

The acceptance of this anatomical condition led to the clinical agreement that when in peripheral facial palsy the lesion was located above the geniculate ganglion there ought to be paralysis of one side of the soft palate demonstrable by relaxation and inability to raise the palate upon the paralyzed side, with a deviation of the uvula to the opposite one.

In 1873 Flint[13] summarized the state of knowledge of that time as follows: "In view of the pathological examples of paralysis of the palate and uvula in certain cases of facial palsy, the frequent occurrence of contraction of the muscles of those parts upon galvanization of the facial, and the reflex action through the glossopharyngeal and the facial, there can be little doubt that the muscles of the palate and uvula are animated from filaments of the seventh nerve."

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[9] Oppenheim, H. Lehrbuch der Nervenkrankheiten, 1905, Vol., p. 405.

[10] Barkas, W. G. Lancet, Jan. 26, 1896, p. 217.

[11] Debron. Thèse de Paris, 1842.

[12] Longet. Traité de physiologie, 1866, Vol. I., p. 796.

[13] Flint, A., Jr. The Physiology of Man, Vol. on the Nervous System, N. Y., 1873, pp. 159 *et seq.*

Later Erb, Seeligmuller, Leube, and Chevostek lent the weight of their authority to the faciopalatal innervation theory, and until 1893 this theory met with but scant opposition. In that year Gowers[14], writing of facial palsy, said: "It has been said that the palate is sometimes paralyzed upon the same side as the face from disease of the facial nerve," etc. * * * "*but the opinion that the palate ever suffers from disease of the facial nerve seems to be erroneous.* The levator is supplied from the spinal accessory and in more than a hundred cases of facial paralysis due to disease of the nerve in various situations, carefully examined, I have never observed a corresponding defect of movement in the palate."

Later Hughlings Jackson[15], in connection with a case of paralysis of the face and palate which seemed to prove the association between peripheral facial paralysis and palatal involvement, but in which closer study demonstrated the cerebral origin of the facial palsy, said: "The uninstructed might have mistaken the case for one of Bell's palsy with paralysis of the palate, a combination of symptoms which I (Jackson) have never met with."

More recently Koester[16], examining thirty-three cases of facial palsy, could find no case with implication of the soft palate, so that he also is a sceptic as to the existence of such a combination.

Since then considerable has been written upon the subject, the majority of authors taking a position in support of Gowers, Jackson, Lermoyez[17], and Koester, while others still maintain that palatal involvement may form an integral part of the clinical symptomatology of peripheral affection of the facial nerve.

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[14] Gowers, William. Manual of Diseases of the Nervous System, Vol. II., 1893, p. 236.

[15] Jackson, Hughlings. Lancet, April 2, 1887, p. 680.

[16] Koester, George. Deutsches Archiv für klinische Medicin, 1900, Vol. LXVIII., pp. 343 and 505.

[17] Lermoyez. Presse médicale, May 7, 1898, No. 39.

Maum[18], of Dresden, writing as late as 1904, deplores the tendency to consider palatal palsy occurring in peripheral facial paralysis as being due to vagoaccessory affections, and, publishing four cases of such a combination due to acute middle ear disease, stoutly maintains that both symptoms are dependent upon an affection of one and the same nerve. He maintains that the type of palatal palsy occurring in vagus affection differs clinically from that due to facial nerve affection.

Others yet take a middle path and assert that, while palatal involvement does occur (Tomke[19]) with more or less frequency in peripheral facial palsy, it is never due to lesion of the facial, but is a result of contemporaneous affection of other nerves (the spinal accessory or its palatine branch). The arguments which have been adduced on the one side or the other are as follows:

1. From the point of view of experiments in physiology.

a. Excitation of the facial.—Béclard[20] (in 1866), Rad, Debron, and more recently Chauveau (experimenting on the horse), Beevor and Horsley (from experiments on various animals), Vulpian[22] (experimenting on the dog), and others are agreed upon the fact that electrical excitation of the facial muscles causes no movement whatsoever in the palate.

The experimental investigations of Rethi[23] have shown that the levator palati mollis is not supplied by the facial, but by the vagus, and Koester, experimenting upon animals, was never successful in observing a paralysis of the soft palate after destruction of the facial nerve as high up as the geniculate ganglion.

b. On the other hand, excitation of the tenth and eleventh pairs produces energetic contraction of the palate.

All these experimental investigations and a case of traumatism (stab wound in a man, Hoffmann[24]) show more and more conclusively that the vagoaccessory carries the chief if not the entire part of the innervation of the palate.

2. Clinical. Here the two questions must be answered.

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[18] Maum, Max. Zeitschrift für Ohrenheilkunde, Vol. XLVII., 1904, pp. 1 to 39.

[19] Tomke. Archiv f. Ohrenheilkunde, Vol. XLIX., 1900.

[20] Béclard. Traité de physiologie, 1866, p. 1010.

[21] Beevor and Horsley. Proc. of the Royal Soc., May 16, 1888.

[22] Vulpian. Acad. des sciences, Oct. 18, 1886.

[23] Rethi. Wiener med. Presse, 1893, No. 50.

[24] Hoffmann, J. D. Zeitschrift f. Nervenheilkunde, Vol. V., 1894, p. 72.

a. Do lesions of the vagoaccessory produce palatal paralysis?

b. Do lesions of the facial produce such paralysis?

The answers to *a* are unanimous and conclusive, viz., paralysis of the soft palate on one side and recurrent laryngeal paralysis upon the same side, without facial palsy, are of not infrequent occurrence. Cases are known in which paralysis of the palate and larynx was accompanied by paralysis of the sternocleidomastoid without being accompanied by facial palsy.

The answers to *b* are more divergent, being affirmative (for competent observers have recorded cases of facial paralysis in which the levator palati and azygos uvulæ were more or less completely paralyzed) as well as negative. An analysis of the affirmative cases, however, shows that certain objections have not been met by the reporters of these cases, viz.: A certain immobility of the palate is physiologically not infrequent. Local affections of the naso-pharynx which may give rise to partial or complete paralysis of the palate have not always been excluded. Congenital asymmetry may be mistaken for unilateral palsy. Sufficient care has not been taken to search for symptoms pointing to a vagoaccessory lesion in addition to the facial one.

On the other hand, it must seem strange that the mere clinical association of facial and palatal paralysis, leaving aside for the nonce the physiological relationship, should have been so infrequently observed by certain clinicians, while others have been able to note its occurrence repeatedly.

It has seemed to me that the explanation of these variations of observation must be sought in the period of time at which the cases have been examined, that cases examined very early in their course will be the ones which may show a palatal involvement, while those cases in which the facial paralysis has existed for some time will certainly show none. We understand, since Broadbent has explained it to us, that those muscles of the body which are used only in conjunction with their fellows of the opposite side have a bilateral cortical representation and can be excited to action from either hemisphere,that these same muscles are equally innervated, or nearly so, through the bulbospinal centers from each hemisphere, the impressions being conveyed freely in either direction through the commissures; and that destruction of the cerebral center of one hemisphere does not cause paralysis of those muscles which are completely bilateral in their actions.

This is well shown when the respiratory muscles, those of phonation, and the muscles of the trunk or the abdominal muscles are involved in consequence of unilateral lesion.

As I have shown elsewhere, this law applies in a way also to peripheral palsies of the same muscles, only here there exists in the beginning a complete paralysis, which in a comparatively short period of time may be overcome by an interposition of the unaffected contralateral hemisphere, so that a certain amount of function or complete function is re-established at a time when other muscles supplied by the affected nerve are still paralyzed or paretic.

Synergic action of both palatal halves under all circumstances shows that they are excitable from either hemisphere, and that therefore if one side of the palate is paralyzed, the excitation could still take place from the opposite hemisphere and the unilateral palsy thus be minimized or perhaps overcome. It has therefore occurred to me that herein we perhaps not only need the explanation of why paralysis of the palate was infrequently or never found in facial paralysis, but also why cases of diplegia facialis, both paths from the cortex being involved, should show a persisting palatal involvement, one which clinically would keep pace with the paralysis of the rest of the muscles affected, provided the palatal muscles were innervated by the facial nerve.

A study of the published cases of facial diplegia from the point of view of palatal involvement is not very satisfactory, for in many of the cases no reference is made to the condition of the palate, and in others it is stated that speech was "nasal in character" and that "no paralysis of the palate existed," two statements which can be reconciled only with difficulty.

In a general way it is stated by Rosenthal[26] that "speech is nasal," by Grasset[27] that "the voice is nasal, liquids pass through the nose," and by Turner that "in complete bilateral facial palsy there is no palatal movement on using the vowel sound "ah,"

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[25] Jacoby, George W. Sign of the Obicularis, Etc. JOURNAL OF NERVOUS AND MENTAL DISEASE, October, 1903.

[26] Rosenthal, M. Lehrbuch der Nervenkrankheiten. Erlangen, 1870, p. 487.

[27] Grasset. Maladies du système nerveux.

and the pronounciation of words requiring the closure of the vaso-pharynx is rendered imperfect, so that "rub" is pronounced "run" and "egg" "eng."

Davaine speaks of the "nasal tone and the paralysis of the velum palati which causes it," and says, furthermore, "when both facial nerves are paralyzed, the velum palati cannot be moved, cannot be lifted, in consequence of which air passes into the posterior nares and thus the nasal tone is produced, and fluids and mucus easily find their way into the nasal passage.

On the other hand Sainton says that "in peripheral diplegia the soft palate is usually intact," and, "of twenty cases collected by Wachsmuth in 1864, in eighteen the palate was unaffected while, of thirteen cases published by eleven different observers since then, which I have analyzed, in only two was such paralysis described."

This infrequent association of palatal palsy and facial diplegia, occurring only four times in thirty-three cases, certainly does not lend support to the facial innervation theory. It is strange that, of the following four cases which I have personally observed, three should show palatal palsy, yet their careful study evinces that here also this palsy is explicable in another way, and that they therefore also speak against the above-mentioned theory.

My cases were as follows::

Case I.—B. S., æt., nineteen, seamstress, came under observation October 28, 1901; she had been perfectly well until October 15; then complained of general malaise, had pains in the extremities and especially in the right side of her face and in front of the meatus of the right ear. This condition continued until the 23d, when she noticed that she could not close the right eye as well as the left, and when she arose the next morning (the 24th) her face was drawn to the left side. This condition remained unaltered during that day, but when she awoke on the 25th her face *was straight again*, but she was unable to move her mouth from side to side and she was told that her eyes did not close. She also had some difficulty in swallowing and complained of pain in front of the meatus of each ear. In contrast to the distortion observed in cases of unilateral facial palsy, the patient attracted attention on account of the marked smoothness of the entire face and on account of its complete immobility and mimic rigidity.

The cheeks and angles of the mouth were flattened and the eyes could not be closed completely and showed the usual rolling upward of the eyeball when closure of the lids was forcibly attempted. She could not pucker up her mouth to whistle, nor could she blow out a candle or retain fluids in the anterior part of her mouth. She seemed to secrete a great deal of saliva, which was constantly dribbling from her mouth. The lips were slightly separated from each other and could not be approximated.

There was some difficulty in chewing (due to weakness of the buccinator), but none in swallowing. Very noticeable was the inability to pronounce any labial letter, b, p, m, v, and f being supplanted by d, t, n, and th. Otherwise speech was in nowise interfered with except that it was decidedly nasal in tone. The tongue could be protruded perfectly straight.

Examination of the fauces showed the uvula to be relaxed in the median line, with its point resting upon the base of the tongue; there was no asymmetry of the arcades of the palate, but the *velum pālati hung relaxed and motionless and did not move upon deep inspiration, upon intonation or upon tickling. At* the same time an almost complete insensitiveness of the palate was noticeable, the patient saying that she felt no touch there; very striking was the ease with which she bore all pharyngeal examinations; the tonsils, the fauces, and the base of the tongue could be freely touched without causing any retching or contraction. Her hearing was good, there was supersensitiveness to noises, and there was no disorder of taste. An electrical examination subsequently made showed reduction of electric excitability on both sides, especially marked in parts innervated by the lower facial. The palatal musculature was not tested.

The course of the case was the usual one of a facial palsy of medium severity, the improvement being steady, but one side, the right, always being somewhat in advance of the left. Ten weeks after the onset complete recovery had taken place. The palatal paralysis was noted at each examination until the fourth week, when, the patient not having been seen for a week, the palate was found to react perfectly upon phonation, *but it did not react reflexly and the entire fauces presented the same insensitiveness to manipulation which has been described.* During the entire course of the trouble no other symptoms than those noted were present—no aphonia, no palpitation, no respiratory difficulty.

Remarks.—This case certainly seems to be one which could be used in support of the faciopalatal innervation theory, as have been those already referred to and published by other observers.

Now, leaving aside all purely anatomical considerations, with the necessary deduction that this case is one with lesions of the facial nerve at or above the geniculate ganglion, we note that the patient presented certain sensory symptoms which persisted and were still present after the palatal paralysis had passed away. Sensory symptoms have been noted in cases published by others (Stintzing[28]), and it is stated that the palate acted upon phonation, but not reflexly. Whatever value such symptoms may possess from a diagnostic point of view, this value certainly does not lie in the line of support of the faciopalatal innervation theory. In view of our knowledge that sensory fibers from the glossopharyngeus and trigeminus probably reach the palate, and certainly do reach the tonsils, posterior pillars of the fauces, and posterior parts of the tongue, while it is just as certain that the facial takes no part in this sensory innervation, we must conclude that in this case there were symptoms present which point to the involvement of other nerves than the facial, and that therefore the vagoaccessory may also have been involved and have been the cause of the palatal palsy.

CASE II.—Mrs. K., æt., thirty, seen in consultation June 16, 1905. She had never been sick. She has four children, the last born about ten days ago. The labor was a perfectly normal one without rise of temperature. On the third day she complained of pains in the right leg and of numbness in the great toe of the right foot. On the following day the other toes of that foot and the toes of the left one became numb. She then had pains all over the body, and on the fifth day numbness of the fingers and of the legs set in and the speech became nasal in character.

On the sixth day she was attacked with left-sided facial paralysis, and two days later the right side became similarly affected. At this time she also complained of buzzing and roaring with pain in both ears.

Examination shows a complete bilateral facial palsy. She cannot close either eye completely, but closes the right eye better than the left. She cannot wrinkle her brows or corrugate the forehead, cannot pucker her mouth, whistle, or blow out a light. The complete paralysis of the upper lip is apparent, and she cannot pronounce a single labial letter, but linguals are pronounced perfectly. Her face is entirely expressionless and gives no evidence of emotion. According to her statement she is no longer able to cry, and *lachrymation has ceased entirely since the attack; no tears are secreted.* Her laugh is merely an inward grunt, and certainly here the words of Davaine are applicable, that "the face seems like a lifeless mask behind which the patient laughs or cries." The masseters were not involved.

The tongue was protruded straight and was freely movable in every direction. Taste was unaffected. *The palate reacted but imperfectly* to direct (reflex) and to indirect (voluntary) stimulation.

During the course of that day her speech became more decidedly nasal in tone and fluids returned through her nose upon attempts at swallowing. She also had paroxysmal attacks of palpitation. On the following day (the 17th) *complete palatal paralysis was present.*. Hearing was overacute in both ears, and she was supersensitive to slight noises.

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[28] Stintzing. Münchener med. Wochenschrift, January, 1893, pp. 1 and 2.

An electrical examination showed reaction of degeneration in all the facial muscles; mechanical excitability of these muscles not increased. Marked tenderness existed in the arms all over the nerve trunks; she could not button her clothes easily, and with her eyes covered she did not know whether she had dropped objects from her hands or not. There was no muscular paralysis, and there was no atrophy. Both knee jerks and both foot jerks were absent, and an extensor paresis of both feet was observable. This condition remained unaltered until the 24th, when she was attacked with lobar pneumonia and died on the third day, with complete consolidation of one lung.

Remarks.—This case is clearly one of multiple neuritis with facial diplegia and palatal involvement. The attacks of palpitation and the difficulty in swallowing indicate that the vagus was involved. The clinical deduction that the paralysis of the soft palate here was a vagoaccessory symptom has quite as much to support it as the assumption that this symptom was due to affection of the facial nerves.

Case III.—Seen in hospital service. Mr. S., æt., forty, November, 1896. There was a marked alcoholic history. The patient came into the hospital suffering from a multiple neuritis. There was drop foot on each side, with tenderness over the nerve trunks of both lower extremities, knee jerks, foot jerks, and sensory disturbances were absent in the legs. The upper extremities showed no objective symptoms, but the patient complained of paresthesia in the fingers of both hands.

In addition to these symptoms, persistent tachycardia was present; the pulse never went below 108, and paroxysmally rose to 180.

On the eighth day after his admission the patient had facial palsy of the left side with disorder of taste; two days later a similar paralysis set in upon the right side. The face then was perfectly smooth and almost devoid of wrinkles.

Chewing and swallowing were attended with difficulty, and here also the characteristic symptom of facial diplegia, the inability to utter any labial letter, was present.

On the following day his speech became nasal and he showed *unilateral palsy of the soft palate*, the palate being relaxed and immobile on phonation, but the relaxation and immobility being confined to the right side; the uvula was distinctly turned toward the paralyzed side. At the same time his voice became hoarse and rough, stridor upon deep inspiration was present, and he had slight difficulty in swallowing. Laryngoscopic examination revealed the right vocal cord standing in the median position and not taking any part in phonation and respiration.

The tongue could be protruded straight.

Ten days later, the clinical picture remaining about the same, an electrical examination showed partial reaction of degeneration in the lower facial territory of both sides and distinct reaction of degeneration in the right side of the palate.

This patient was seen at intervals for about three months, during which time all the symptoms, except the palatal palsy and the vocal cord paralysis, disappeared. These later symptoms persisted at the time of his discharge from the hospital.

Remarks.—We are here dealing with a multiple neuritis of alcoholic origin in which numerous symptoms of vagus involvement are present. These symptoms are aphonia, palpitation, and respiratory difficulty. Cases of palatal palsy with recurrent paralysis are not unknown, and a form of vagus neuritis limited to one recurrens is occasionally encountered. In multiple neuritis, especially the alcoholic form, bilateral facial palsy is not unusual (Starr), nor is vagus neuritis infrequently met with (Mays).

The simultaneous occurrence of bilateral facial palsy and unilateral recurrens paralysis has, so far as I know, not previously been described; that the palatal palsy here is also due to vagus involvement is, I think, proved by the fact that it, as well as the recurrens paralysis, is unilateral and both are upon one and the same side, while the facial palsy is bilateral. Of interest also is the degenerative change found on the right side of the palate, showing that the vagus complication was also a neuritic one.

Such electrical changes in the palate have been found by Hill[29].

CASE IV.—Bilateral facial palsy without palatal involvement. A woman, æt. thirty-four, had pains in both ears for several days, when, in October, 1904, she awoke with facial palsy of the right side, especially noticeable on talking and chewing. Three days later the left side became affected. There was a marked affection of taste, but no dryness of the mouth; taste was lost on the anterior two-thirds of the tongue, the patient being unable to distinguish salt from sugar or vinegar from a solution of quinine. The ears were very sensitive to noises; sensation everywhere in the face was unaffected. There were no changes of electric excitability in the muscles or nerves.

Speech was not nasal in tone, but the interference with the formation of labials was the same as in my other cases. The palate responded perfectly to all forms of stimulation.

During sleep the lids, which otherwise could not be closed, were gradually and completely approximated, so that the entire eyeball became covered.

Recovery took place in about eight weeks.

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[29] Hill. British Med. Journal, Feb. 2, 1899.

General remarks.—It has been stated that paralysis of the dilatatores narium and the compressores narium renders the alæ flaccid, so that in breathing they move to and fro and close the nostrils upon deep inspiration. Thus, in the case of Labadie Lagrave[30] and Emile Boix it is stated that the alæ nasi were passive and, instead of dilating at each inspiration, had a tendency to close the nares. This was especially noticeable in an effort to smell anything, such as cologne.

Were this a fact, then another explanation than that of weakness of the soft palate could hereby be given for the nasal tone which at times accompanies bilateral facial palsy, but in none of my cases could this statement be made, and the nostrils were neither dilated nor contracted, but remained patent throughout, being merely somewhat narrowed. It is indeed difficult to believe that this could ever be otherwise, for the inner and outer walls of each nostril are formed by lateral cartilages, so connected to the upper cartilages and the front part of the cartilage of the septum, as well as with the nasal process of the superior maxilla, that inactivity of the muscles which act upon them can never cause them to collapse.

The nasal tone is therefore always due to weakness of the velum palati, and when this symptom is present, and it has been present when the palatal movements were not decidedly interfered with during phonation, the conclusion is warranted that paresis of the palate exists. This speech disorder due to palatal weakness, being added to that caused by the labial paralysis, greatly emphasizes the characteristic speech difficulty.

Reports differ as to the position of the uvula in unilateral facial palsy, some observers saying that it is turned toward the paralyzed, others that it is turned toward the sound side, so that as far back as 1853 Debron looked upon the deviation of the uvula as an accidental occurrence in health, having no concern with the facial paralysis, and Bernhardt, in 1876, refused to draw conclusions as to the involvement of the palate from such deviations in posture. To-day the complete symptomatic insignificance of such deviation is accepted.

So also in bilateral facial palsy it would be unwarranted to infer palatal weakness from deviation of the uvula.

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[30] Archives générales de méd., 1896. Vol. I., p. 23.

In all of my cases the tongue could be protruded straight, thus showing what has been demonstrated by Hitzing[31] and since accepted by every one, that the tongue does not deviate in facial paralysis, but that the apparent deviation is due to crookedness of the face and can be corrected by straightening the mouth.

In bilateral facial palsy, there being no distortion of the mouth, the tongue is not protruded to one side.

In all of my cases the facial palsy of the two sides came on at intervals, first one and then the other side being affected. This has been so also in all reported cases, and in one of Hoffmann's[32] the palsies came on a month apart.

The classical symptom of absolute inability to utter any labial letter was present in all my cases, and in all of them it was of interest to observe how little interference with such pronunciation the unilateral palsy caused, and how marked it became when the other side became paralyzed.

In conclusion, and referring again to palatal palsy as a concomitant of bilateral facial paralysis, I would call attention to the necessity of seeking for symptoms which cannot be explained by facial nerve involvement alone, such as palpitation, sensory disturbances, hoarseness, etc. Whenever such symptoms are found to be present persistently, the conclusion is warranted that some other nerve than the facial has been implicated by the morbid process and that the co-existent palatal palsy must be due to such implication.

This conclusion agrees entirely with what we know of the associated functions of the vocal apparatus, for, as Lermoyez[33] and Rousseau[34] have so well said: "Is it not more logical to admit that the larynx and pharynx (the reed and the resonator), which are after all but two parts of the same vocal apparatus, should respond to the influence of one and the same nerve? And would it not be difficult to understand that the larynx and soft palate, destined to live in accord, should receive their orders from two nerves so foreign to each other as are the facial and the vago-accessory?"

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[31] Hitzing. Gesellschaft für Psychiatrie, Nov. 14, 1892.

[32] Hoffman, J. Deutsche Zeitschrift für Nervenheilkunde, Vol. V., 1894, p. 72.

[33] Lermoyez, L. Presse méd., May 7, 1898, No. 39.

[34] Rousseau, L. Thèse de Paris, No. 503; also Gaz. hebd. de méd et de chirurg., 1898, p. 1009.